allowed to increase during the induction or the maintenance of anesthesia, but how much it should be reduced is controversial. In this regard, the two major types of anesthetics, inhaled volatile agents and narcotics, differ significantly.

Use of the inhaled volatile anesthetic halothane (Fluothane) results in a dose-related depression of myocardial contractility, cardiac output and mean arterial blood pressure. Heart rate and systemic vascular resistance remain unchanged. Myocardial blood flow and oxygen extraction decrease with an appropriate increase in coronary vascular resistance, implying normal autoregulation of the coronary circulation. This reduction in myocardial work and oxygen consumption has been termed "low pressure-low demand" anesthesia. Undesirable effects, however, including possible junctional and ventricular dysrhythmias, as well as significant biotransformation, have tempered its use.

Isoflurane (Forane), a newer volatile anesthetic with little biotransformation, is now in widespread use. Data from healthy volunteers have suggested that it preserves myocardial contractility. In older patients undergoing a surgical procedure, however, its direct effects are similar to those of halothane. Systemic vasodilation results in a lowering of vascular resistance that "unloads" the heart, reducing myocardial wall tension. A decrease in the mean arterial blood pressure may occur due to its negative inotropic effects. The heart rate may increase and require a narcotic or a β-blocker for control. Reiz and co-workers noted increased coronary blood flow and coronary sinus oxygen content associated with reduced coronary vascular resistance, suggesting direct vasodilation of the coronary circulation with "luxury perfusion." Several of these patients showed lactate production, electrocardiographic evidence of ischemia or both, which suggested a "coronary steal" phenomenon. Further studies are needed, especially those dealing with outcome. Enflurane (Ethrane), which behaves like halothane in many respects, may also be a coronary vasodilator (although to a lesser degree than isoflurane).

The narcotic anesthetics have gained popularity since a lack of significant cardiac depression with the use of high-dose morphine was documented. The synthetic narcotic fentanyl citrate has greater potency, more rapid onset and elimination and it lacks histamine release. Studies have documented its lack of myocardial depression and the absence of direct effects on the coronary circulation. In addition, the heart rate is usually unchanged or even lowered, which will prolong diastolic coronary perfusion. Sufentanil citrate (Sufenta), a new, more potent analog of fentanyl now in clinical use, has cardiac effects similar to those of fentanyl. The principal problem with the use of high-dose narcotics as sole anesthetics is an incomplete attenuation of autonomic responses to surgical stimulation, causing hypertension and tachycardia with resultant increases in myocardial oxygen consumption and lactate production. However, used in combination with other intravenous agents such as diazepam, β-blockers, vasodilators or with volatile anesthetics, these responses may be controlled.

Although no sole anesthetic agent is ideal, the use of a combination of a narcotic and a volatile anesthetic may be best in a patient with an ischemic, nonfailing ventricle. Data on the use of volatile agents in patients with a failing ischemic ventricle are limited; however, the potential for a pronounced reduction in coronary perfusion pressure exists. No study has shown the superiority of any anesthetic in patients with myocardial ischemia. Indeed, the most important variables will remain the well-controlled induction, maintenance and emergence from anesthesia using appropriate electrocardiographic, hemodynamic and, possibly, echocardiographic monitoring. A key aim should be to maintain an adequate coronary perfusion pressure while avoiding increases in heart rate or afterload. Ischemia should be treated aggressively, either by manipulating the anesthetic level or by administering nitrates or calcium channel or β-blockers.

MARTIN J. LONDON, MD
San Francisco

REFERENCES

Curling PE, Kaplan JA: Inhalation: Effects of anesthetics on the coronary circulation, *In* Kaplan JA (Ed): Cardiovascular Pharmacology—Vol 2, Cardiac Anesthesia. New York, Grune & Stratton, 1983, pp 123-141

Reiz S, Balfors E, Sorensen MB, et al: Isoflurane—A powerful coronary vasodilator in patients with coronary artery disease. Anesthesiology 1983 Aug; 59:91-97

Slogoff S, Keats AS: Does perioperative myocardial ischemia lead to postoperative myocardial infarction? Anesthesiology 1985 Feb; 62:107-114

Sonntag H, Larsen R, Hilfiker O, et al: Myocardial blood flow and oxygen consumption during high-dose fentanyl anesthesia in patients with coronary artery disease. Anesthesiology 1982 Jun; 56:417-422

Patient-Controlled Analgesia—A New Concept in Postoperative Pain Control

CONTROLLING postoperative pain is a problem for patients and is often a frustration for health care personnel. Physicians' and nurses' fear of opiate side effects leads to the underprescribing of analgesics both in terms of dose and dosing interval: too little is given too seldom. This problem is made worse by wide individual patient variation in analgesic requirements. The search continues for the perfect analgesic (pain control with no side effects), but even if this highly sought agent is found, pain relief will likely be limited by the method of administration. Ideally, an analgesic is given to achieve a uniform serum concentration tailored to a patient's requirements for pain relief, yet using the minimal dose to keep side effects acceptable. Patient-controlled analgesia is a new way to administer analgesics that meets these requirements.

Patient-controlled analgesia (PCA) is a method by which a patient intravenously self-administers small doses of opiate to meet his or her analgesic needs. A computer-controlled syringe pump is programmed for an analgesic dose and minimal dosing frequency, and the patient triggers delivery by the push of a button. Thus, the patient can achieve an ideal balance between analgesia and analgesic side effects. With small incremental doses of opiate, serum concentrations are relatively constant and change with the patient's needs. The equipment for this technique has only recently become available; the Harvard PCA model (C.R. Bard, Inc) is the most versatile and sophisticated apparatus. Morphine sulfate has been most frequently used with this technique in doses of 1 to 3 mg and with a minimal dosing frequency of 6 to 15 minutes, the shorter intervals with the smaller doses. The higher doses, although perhaps more convenient for a patient, achieve higher peak blood concentrations and may more frequently cause side effects. Other medications have been used, but none have proved superior to morphine.

As a new technique, PCA must be compared with commonly used methods for analgesic dosing. It is superior to

morphine given intramuscularly on an "as-needed" schedule; PCA causes less sedation or nausea and gives more consistent pain relief. An opiate given intramuscularly on a regular schedule compares more favorably with PCA, with both techniques offering considerable improvement over analgesia given as needed. Less information is available on continuous intravenous infusion of morphine, but PCA appears to be superior. With PCA, serum levels may be altered by patients as their needs change, but with a continuous infusion, a delay is introduced because the patients depend on a nurse or physician to modify the infusion for improved analgesia or reduced side effects. Patient participation and control may be important in the effectiveness of PCA. Reduced anxiety levels and a placebo effect may improve analgesia. Not only have serious side effects with PCA been rare, but it may shorten hospital stays because respiratory function and possibly postoperative ambulation are improved.

Although PCA has wide applications, there are some limitations to its use. Patients must be able and willing to participate in their own care and must understand the general principles of the concept. This technique should be avoided in patients with a narcotic-abuse history because they might have difficulty separating pain relief from the other effects of the opiate medication, such as euphoria. On the other hand, in patients in whom tolerance to opiates has developed but who are not at risk for abuse, this technique could allow the patients to compensate for their increased requirements for medication.

In conclusion, patient-controlled analgesia appears to be a safe and superior method for postoperative pain control. At present morphine appears to be the analgesic of choice. With this technique, most patients will have good analgesia with minimal side effects.

BRADFORD D. HARE, MD, PhD
Salt Lake City

REFERENCES

Bennett RL, Batenhorst RL, Foster TS, et al: Postoperative pulmonary function with patient-controlled analgesia (Abstr). Anesth Analg 1982 Feb; 61:171

Graves DA, Foster TS, Batenhorst RL, et al: Patient-controlled analgesia. Ann Intern Med 1983 Sep; 99:360-366

Klieman R, Lipman A, Hare B, et al: Controlled study of patient controlled analgesia in severe post-operative pain. Pain 1984; (suppl 2):S387

Spinal Narcotics

ONE OF THE MOST exciting and clinically important recent advances in anesthesiology has been the treatment of pain by injecting narcotics into the subarachnoid or epidural spaces (spinal narcotics). Spinal narcotics are effective in managing cancer and chronic back pain, causalgia, claudication and pain associated with myocardial infarction, thrombophlebitis, herpes zoster and nephrolithiasis. Because spinal narcotics relieve both visceral and somatic pain, they provide intense, long-lasting pain relief following all types of surgical procedures.

Spinal narcotics produce a selective neuronal block—that is, only the sensation of pain is affected. Thus, their major advantage over local anesthetics is the complete avoidance of motor and autonomic nervous system blockade. Patients treated with spinal narcotics are comfortable, can breathe deeply, cough and ambulate earlier. This in turn reduces the risks of pulmonary emboli and other respiratory tract complications.

Respiratory depression, a complication of spinal narcotics, is encountered more frequently after subarachnoid administration. It is due to the rostral spread of narcotic through the cerebrospinal fluid to the brain stem. Respiratory tract problems usually occur within the first 6 hours but can occur as late as 24 hours after injection. Although sudden apnea has been reported, a gradual slowing of the respiratory rate or a decrease in tidal volume is more common. Respiratory tract complications can be reversed immediately with intravenous administration of naloxone hydrochloride without affecting analgesia.

Clinically significant respiratory depression is rare in patients previously made tolerant to narcotics. Thus, there have been no reports of respiratory arrest in patients with chronic cancer pain treated with morphine sulfate who subsequently receive large amounts of spinal narcotics. Even for patients having a routine surgical procedure who are treated with epidural narcotics, respiratory tract complications are infrequent and usually occur only when parenteral narcotics are also given.

Although respiratory depression is uncommon, all patients receiving spinal narcotics should be observed closely. In a recent survey, 18% of American anesthesia departments reported that at their institutions spinal narcotics were routinely administered in surgical wards. Many anesthesiologists, however, still prefer to use spinal narcotics only in an intensive care unit or a postanesthesia recovery room where their patients' respiratory state can be monitored.

A unique and relatively minor side effect of spinal narcotics is pruritus. Urinary retention is also common. Histologic examination of spinal cord specimens from patients with cancer treated with epidural morphine as long as six months showed no evidence of neurologic damage. Spinal narcotics have never been associated with neurotoxicity, but as a safety precaution only preservative-free solutions are used.

With increasing lipid solubility, potency is increased. Lipophilic narcotics, however, have a shorter effective duration of action (Table 1). For example, less hydromorphone hydrochloride than morphine is needed for an equivalent block

TABLE 1.—*Epidural Narcotic Dosage, Onset and Duration of Action*

Drug	Dose, mg	Complete Pain Relief, min	Analgesia Duration, hr
Fentanyl citrate (Sublimaze)	0.05 to 0.1	20	4.0
Hydromorphone hydrochloride (Dilaudid)	1.0 to 1.5	25	11.5
Meperidine hydrochloride (Demerol)	30 to 100.0	20	6.0
Methadone (Dolophine) hydrochloride	5.0	17	8.0
Morphine sulfate (Duramorph)	5 to 10.0	45	18.0